ABSTRACT

A growing literature reports traffic exposure impacts on children's respiratory health, yet few U.S. studies have used advanced Geographic Information System modeling techniques to estimate exposures on a fine spatial scale. We developed a land use regression (LUR) model to estimate long-term exposure to traffic air pollution for 1,387 children who participated in the Los Angeles Family and Neighborhood Wave Two Survey (L.A. FANS-2). Using passive badges, we conducted two-week measurements of NO_x and NO₂ at approximately 200 sites in two seasons in 65 neighborhoods and built LUR prediction surfaces on a 25 x 25 meter grid over the L.A. Basin that explained 81%, 86% and 85% of the variation in NO, NO₂ and NO_x concentrations, respectively. Annual average concentrations at geocoded L.A. FANS-2 residential and school locations were extracted from the LUR surfaces and weighted by time spent at each location for various exposure periods (current home, 1-year, 2-years, 5-years prior to interview). Exposure surfaces for O₃ and PM_{2.5} were generated by kriging available government monitoring data.

Multivariate logistic regression was used to estimate associations between these exposure metrics and doctor-diagnosed asthma (ever), wheeze in the past year ("current wheeze"), and medication use for asthma and wheeze in the past year ("current medication use"). Multivariate linear regression was used to estimate associations with cross-sectional measures of lung function assessed via EasyOneTM portable spirometers. Children more highly exposed to traffic pollution as estimated by LUR models for NO, NO₂ and NO₃ were approximately 30-40% more likely to report current wheeze regardless of adjustment for many family- and neighborhoodlevel socioeconomic factors. Smaller (15%) increases in odds were observed for current medication use and for doctor-diagnosed asthma (per interquartile (IQR) increase in NO, NO₂ and NO_v). In stratified analyses by median census tract-level economic disadvantage, odds for both asthma outcomes in higher SES areas only were found to increase by about 40% per IQR increase in traffic pollution. This may, in part, reflect differential access to health care and resulting differences in asthma diagnosis and reporting. In lower SES areas, we estimated 80-100% increases in odds of current wheeze and medication use per 30 ppb increase in peak daily O₃, while null or inverse associations emerged for peak O₃ in children from higher SES areas, potentially reflecting differences in children's time-activity patterns (e.g., outdoor physical activity) and resulting exposures during O₃ peak hours in higher versus lower SES areas. However, these findings are based on a relatively small sample size in each SES stratum.

We estimated 70-100 mL reductions in lung volume and 60-100 mL/s reductions in expiratory flow per IQR increase in NO, NO₂ and NO_x in boys with one or more acceptable spirometry curves. Smaller associations were observed for PM_{2.5} (40-50 mL reductions in volume and 60-90 mL/s reductions in flow per IQR increase). However, when restricting analyses to boys with three acceptable and reproducible curves, negative associations were less precisely estimated and did not reach statistical significance, except for PM_{2.5} with FEF₇₅ and FEF₂₅₋₇₅. In girls, we estimated even greater associations between traffic pollution and expiratory flow (300-350 mL/s reductions in PEF and 200-300 mL/s reductions in FEF₂₅₋₇₅ per IQR increase in NO, NO₂ and NO_x), but results were not replicated in the group with three acceptable and reproducible curves. We also observed reductions in PEF in girls more highly exposed to peak daily O₃ (~100 mL/s decrement and ~400 mL/s decrement per 30 ppb increase in O₃ for girls with one or more acceptable curves and three acceptable and reproducible curves, respectively). Similar to previous literature, our results suggest important differences in the biological impact of air pollution on lung function in boys versus girls.